Amendments to the Specification:

Please replace paragraph [0020] with the following amended paragraph:

[0020] Figures 6A-D show the transcriptional regulation of RTP801. The human sequence in Figure 6B is SEQ ID NO:12. The mouse sequence in Figure 6B is SEQ ID NO:13.

Please replace paragraph [0035] with the following amended paragraph:

[0035] The present invention further provides proteins and their analogues as encoded by the nucleic acid sequences as set forth in SEQ ID NO:1, SEQ ID NO:2, SEQ ID NO:3, SEQ ID NO:4, and SEQ ID NO:5, with SEQ ID NOs:7-11 ~~and 8 as well as SEQ ID NOs:9-11~~ being exemplars of the proteins. The present invention further provides a method of regulating angiogenesis or apoptosis in a patient in need of such treatment by administering to a patient a therapeutically effective amount of a protein encoded by SEQ ID NOs:2-6 or the protein sequences as set forth in ~~SEQ ID 8,10-11~~SEQ ID NOs:7, 8, 10-11 as active ingredients in a pharmaceutically acceptable carrier.

Please replace paragraph [0056] with the following amended paragraph:

[0056] Many reviews have covered the main aspects of antisense (AS) technology and its enormous therapeutic

potential (Wright and Anazodo, 1995). There are reviews on the chemical (Crooke, 1995; Uhlmann et al, 1990), cellular (Wagner, 1994) and therapeutic (Hanania, et al, 1995; Scanlon, et al, 1995; Gewirtz, 1993) aspects of this rapidly developing technology. Isolation of inhibitory antisense RNA is disclosed in Holzmayer (1992). Within a relatively short time, ample information has accumulated about the *in vitro* use of AS nucleotide sequences in cultured primary cells and cell lines as well as for *in vivo* administration of such nucleotide sequences for suppressing specific processes and changing body functions in a transient manner. Further, enough experience is now available *in vitro* and *in vivo* in animal models and human clinical trials to predict human efficacy.

Please replace paragraph [0059] with the following amended paragraph:

[0059] Phosphorothioate antisense oligonucleotides do not normally show significant toxicity at concentrations that are effective and exhibit sufficient pharmacodynamic half-lives in animals (Agrawal et al, 1996) and are nuclease resistant. Antisense induced loss-of-function phenotypes related with cellular development were shown for the glial fibrillary acidic protein (GFAP), for the establishment of tectal plate formation in chick (Galileo et al, 1991) and for the N-myc protein, responsible for the maintenance of cellular

heterogeneity in neuroectodermal cultures (epithelial vs. neuroblastic cells, which differ in their colony forming abilities, tumorigenicity and adherence) (Rosolen et al, 1990; Whitesell et al, 1991). In the latter publication the antisense oligonucleotide is antisense RNA. Antisense oligonucleotide inhibition of basic fibroblast growth factor (bFgF), having mitogenic and angiogenic properties, suppressed 80% of growth in glioma cells (Morrison, 1991) in a saturable and specific manner. Being hydrophobic, antisense oligonucleotides interact well with phospholipid membranes (Akhter et al, 1991). Following their interaction with the cellular plasma membrane, they are actively (or passively) transported into living cells (Loke et al, 1989), in a saturable mechanism predicted to involve specific receptors (Yakubov et al, 1989).

Amendments to the Sequence Listing:

Please enter the attached Sequence Listing, numbered as pages 1-22.

Please substitute the attached Sequence Listing section for the Sequence Listing filed March 6, 2002.

A new computer-readable form is also attached.